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GALLSTONE DISEASE.[1]

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While, of late years, much consideration has been given to the subject of gallstone disease, and while many brilliant cures have been brought about by the proper application of surgical methods, the last word on the subject has yet to be written, and there are many moot points of equal interest to the internist and the surgeon that impress me with the importance of further discussion of disease of the biliary tract. By reason of its function as the excretory apparatus of the largest gland in the body, by reason of its intimate relations with vital organs in the upper abdomen, and by reason of its liability to infection and its commanding position to spread infection over a greater or less extent of the peritoneal cavity, the biliary apparatus may justly be considered unique.

To many surgeons the biliary apparatus consists, surgically speaking, of the hepatic ducts, the gall-bladder, the cystic duct, and the common duct; but studies in the pathology of the living have impressed me with the importance of considering the extent of the biliary tract from its developmental standpoint. Thus, every secreting cell in the liver is surrounded on one or more sides by a bile capillary; these capillaries unite to form bile vessels situated between the lobules of the liver; the bile vessels, increasing in size, unite with one another until eventually the large common hepatic duct is formed, and this, uniting with the cystic duct from the gall-bladder, forms the common bile duct. But not only developmentally, but also clinically, the biliary tract does not end here. Embryologically the pancreas with its main duct and intermediate tubules is derived from the outgrowth of the biliary tract from the duodenum, and clinically this abdominal salivary gland is frequently secondarily involved in gallstone disease. Hence, considered in this wise, there is a very extensive area comprising countless square inches, which, like the mucous membrane of the respiratory and the urinary systems, affords a broad and fertile field for the implantation and unlimited spread of infection.

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[1] Read at the Forty-first Annual Meeting of the Canadian Medical Association, Ottawa, June 11, 1908.

The common infectious agents of the biliary tract, in order of their frequency, are: Bacillus coli communis, Bacillus typhosus, and the common pyogenic organisms. How do these bodies invade the biliary tract? The three ordinary pathways are the portal circulation, the common bile duct, and the systemic circulation.

What is the immediate result of infection of the biliary tract? "As in other mucous canals . . . . the production of a catarrh, with the usual inflammatory phenomena—œdema and congestion of the mucous membrane, increased production of mucus, and desqumation of epithelium. If the biliary circulation is free and unimpeded the results of this catarrh are washed away for the most part, but on account of special local conditions (largely dynamic) they are likely to accumulate, to become accentuated, and to persist in the gall-bladder. In the event of obstruction to the free flow of bile these are the more certain to occur. In many cases the lesions thus provoked are entirely latent or unannounced by noteworthy or unequivocal symptoms; they may pursue a short course, or they may continue for years; and they are one of the most important factors, in fact the important factor, in the etiology of gallstones.

"This infection of the biliary tract is of the utmost significance, and forms an integral part of what is commonly designated calculous cholecystitis and cholangitis. The phenomena may develop: (1) Acutely, without preceding clinical signs referable to the gall-bladder; (2) subacutely; and (3) chronically. The ensuing pathological lesions are of the greatest diversity: Thus, for instance, gallstones may or may not be present, and the concomitant inflammatory phenomena may be of varying grades—from the mildest catarrhal lesions to widespread phlegmonous and ulcerative processes that may lead to perforation or gangrene of the gall-bladder; gallstones, if present, may be quiescent or active; they may be present in the gall-bladder, or in any one of the ducts, or in all the ducts, or in the ducts and not in the gall-bladder; they may cause an acute or chronic, partial or complete, temporary or permanent, obstruction of the cystic, the hepatic, or the common bile ducts, and, on the other hand, such obstruction may occur in the absence of gallstones (being due to swelling of the mucous membrane, kinking of the ducts, or obstruction from without), and in the presence of gallstones the ducts may be partially or completely patulous; the gall-bladder may be distended or contracted, its walls thinned or much thickened, and its lumen ultimately may become almost if not quite obliterated; it may contain bile, mucus, blood, or pus, or combinations of these, in addition to or in the absence of gallstones; adhesions may form between the gall-bladder and adjacent structures (the liver, the stomach, the duodenum, the colon, the omentum, etc.), and by way of the adhesions the gallstones may rupture into the gastro-intestinal tract and sometimes cause intestinal obstruction; or purulent pericholecystitis and pericholangitis, localized or generalized peritonitis, pylephlebitis, pericholangitic abscesses of the liver, fistulæ, acute and chronic pancreatitis, etc., may ensue; and, finally, in some cases a general bacterial, often pyococcic, infection, with or without multiple abscess, may develop."[3] Furthermore, the long-continued infection may seriously involve the arterial and urinary systems, producing such degeneration of the myocardium and kidneys that often makes the surgeon loath to operate.

Cholecystitis may be calculous or non-calculous. The larger one's experience in gallstone disease the less able is he to state whether gallstones are, or are not, associated with cholecystitis. Cholecystitis is caused by invasion of the fertile mucous membrane of the gall-bladder by pathogenic bacteria. This event brings about the classical symptoms of inflammation. Pain is situated in the epigastric region, is usually sharp, appears suddenly or gradually, and is increased by motion either of the entire body or of regional organs, such as the stomach in peristalsis or in vomiting. The accompanying tenderness indicates the situation of the gall-bladder in the absence of peritonitis; the presence and extent of peritoneal irritation or peritonitis is indicated by the extent of the associated tenderness. The swelling of the mucous membrane occludes the cystic duct, and thus isolates the gall-bladder from the intestine. By loss of function the gall-bladder, instead of being the reservoir for bile, now becomes the repository of pathological exudates. This causes distention of the organ, and thus the tumor may, in favorable cases, be palpated. In the case of the gall-bladder, loss of function bears much significance, as the products of inflammation may lead to the formation of gallstones.

The severity of the cholecystitis is indicated by the general symptions, and varies with the virulence of the infection; for example the simple pathological processes of œdema and congestion of the mucous membrane, increased production of mucus, and desquamation of epithelium unite to form the clinical entity of acute catarrhal cholecystitis. This entity makes itself known to the outside world by the general symptoms of fever with its accompanying phenomena of chill, accelerated pulse, vomiting, dry and coated tongue, scanty high-colored urine, and leukocytosis. Locally, there is pain, tenderness, and rigidity. Such a mild case I consider appropriate for judicious medical treatment, for if the biliary circulation is free and unimpeded the results of this catarrh are washed away for the most part. If this mild cholecystitis yields promptly to medical treatment, and the patient is having his first attack, I think operation may well be postponed until further symptoms arise. Calculi are probably absent in such mild cases in early attacks, and such patients seem to me particularly suited for the so-called Carlsbad cure. When the attacks of cholecystitis are repeated, and especially when discomfort persists in the intervals, operation is demanded to obtain a cure. The frequency with which mild but recurrent attacks of cholecystitis give rise to pericystic and pyloric adhesions, I fear, is not duly appreciated by all of the profession.

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[3] A. O. J. Kelly: Infections of the Biliary Tract. Amer. Jour. Med. Sci., 1906, cxxxi, 446.

But suppose the medical attendant does not see the mild case clear up, that the symptoms become more severe: what is going on in the gall-bladder? There is obstruction to the free flow of bile, and as Dr. Kelly says because of special local conditions (largely dynamic) the results of the catarrh accumulate, become accentuated, and accumulate in the gall-bladder. Here the infection is more virulent, and the pathological changes have now produced the clinical entity of acute purulent cholecystitis, or empyema, a condition which I found in 16.4 per cent. of a series of 182 cases recently operated upon. The fever now becomes hectic in type, and the brow is bathed in sweat; there may be delirium of the low, muttering type. Local pain, tenderness, and rigidity, are increased. The patient loses flesh, his appetite is poor, and he frequently asks for water to drink. Here medical means are futile, but a cure is usually effected by cholecystostomy, during which procedure any obstruction that may be found in the cystic duct should be removed. Empyema requires cholescystectomy only when of long standing, such chronicity resulting in irreparable disease of the walls of the gall-bladder.

If the infection be very virulent, the walls of the gall-bladder will become infiltrated by the products of phlegmonous inflammation, which may undermine the mucous membrane, interfere with the blood supply of the organ, and lead to gangrene. Other varieties of acute cholecystitis, of pathological interest, are the hemorrhagic and the ulcerative. The hemorrhagic form is made manifest, when, upon opening the gall-bladder, blood clots are found. In this instance microscopic ulcerations are active in opening into congested veins, and it is possible that, for a time, at least, the bleeding is furthered by the local anti-clotting action of the bile, an action that, to the annoyance of the surgeon, is most manifest in that state known as cholemia. Ulcerative cholecystitis becomes of interest clinically when it results in perforation of the gall-bladder. Gangrene of the gall-bladder naturally requires excision of the gall-bladder, and also does perforation, since it occurs most frequently when the gall-bladder walls are extensively diseased.

If unchecked by surgical means the infection may spread from the gall-bladder through the cystic and hepatic ducts into the interlobular bile vessels, and even into the intercellular bile capillaries. Such a pathological extension creates the clinical entity of infective cholangitis, the symptoms of which are manifested by ague-like attacks, to which Charcot has applied the term of intermittent hepatic fever. To the previous symptoms of cholecystitis there is now added the sign jaundice, which may be of the intermittent type. But it must be remembered that infectious cholangitis often follows upon the impaction of a calculus in the common bile duct. The treatment of this condition is to see that the entire biliary tract is clear and unobstructed, and to secure free drainage.

If unrelieved acute infectious cholangitis may be the forerunner of suppurative cholangitis. Suppurative cholangitis may also arise from acute infectious disease, especially typhoid fever and influenza, from carcinoma of the bile ducts, and from hydatid disease. Symptoms of septic fever, rapid loss of flesh, and obstinate jaundice now supervene. The treatment is that of infectious cholangitis, namely, bile drainage.

Acute purulent hepatitis is the terminal condition of this triumphal march of bacteria from the gall-bladder to the liver. In this organ, wrecked by infection, the last stand of the organism is made: multiple hepatic abscesses form, and the patient finally succumbs from the uncontrollable fever and toxemia. It is in such cases that operation retards the progress of modern surgery. If the infection be less virulent, biliary cirrhosis of the liver, usually the result of gallstone blockage, may ensue. In the presence of this latter entity, clearance and drainage of the biliary tract may relieve the patient by depleting the liver, but it cannot remove the adventitious fibrous hypertrophy.

Having traced infection of the biliary tract by continuity let me trace it by contiguity. It is here that a gall-bladder, its walls permeated by phlegmonous infiltration, becomes a paramount source of danger by reason of its commanding position to spread infection over a greater or less extent of the peritoneal cavity, a circumstance to which attention was directed in the early part of this paper. The virulent bacteria may pass directly through the gall-bladder walls, or give rise to ulceration or perforation, and then quickly involve the neighboring peritoneum. If the peritoneum be alert, pericholecystic abscess may ensue, with marked tenderness and rigidity; a correct diagnosis of this condition should lead to immediate evacuation. In neglected cases the pus, like water, follows in the direction of least resistance, and this may lead it into the peritoneal cavity, the alimentary tube, the subhepatic retroperitoneal tissue, or, if it accumulate between the superior surface of the liver and the right cupola of the diaphragm, it may creep through this into the right pleural sac and the lung, to be spat up by the patient. Much more commonly than this, however (in 58.7 per cent. of 182 cases recently reported by me), the diffusion of the toxins in the regional peritoneum in small repeated doses, evokes patches of fibrinoplastic exudate, which reach out octopus-like for nearby organs, and, by subsequent contraction, throttle more or less completely those that are hollow, as the stomach, duodenum, or colon, thus increasing the complaint of the patient and the operative difficulty of the surgeon. The omentum, the organ of phagocytosis, may complete the Gordian knot by fettering the already shackled tissues to the liver; the frequency with which such masses are mistaken for malignant disease is attested by operators. However, until this tangle of tissue is established, the patient's life probably hangs in the balance, for such a mass of adhesions may represent the only alternative to diffuse purulent peritonitis.

A gall-bladder once damaged by infection may, if the infection be mild, undergo complete resolution. Repeated mild attacks of inflammation, or a severe and extensive infection may determine chronic cholecystitis. Interference with the physiological periodic expulsion of bile from the gall-bladder—a condition brought about by permanent obstruction of the cystic duct from chronic inflammatory changes, or by a stone—results in absorption of the bile contained therein at the time of blockage, and its replacement by clear, watery mucus. Thus hydrops of the gall-bladder occurd in 7.1 per cent. of 182 cases recently reported by me. This condition is one of the few that calls for cholecystectomy, since obliteration of the cystic duct renders the gall-bladder not only useless, but also a continued menace from the liability to reinfection or even of rupture. The only exception I would make to this rule is obstruction of the cystic duct by stone, if recent; if the gall-bladder show but little macroscopic evidence of disease, and if after dislodgement of the stone bile flows immediately into the gall-bladder, it may be wise to leave the organ. Destructive bacterial invasion of the walls of the gall-bladder is followed by the ultimately destructive effects of constructive repair, resulting in abolition of the normal suppleness of this little bile-containing bulb. Contraction, that inevitable consequence of fibrous degeneration, gradually obliterates the cavity of the gall-bladder, and the surgeon sees frequent instances in which this has been accomplished. What space may remain in such an organ is usually filled with ropy, tarry, inspissated, and pathologically altered bile. Such shriveled gall-bladders should be drained or removed, in keeping with the possibilites of the case and the judgment of the operator.

In order to emphasize these remarks by practical illustrations, I have analyzed the 72 cases of gallstone disease operated upon by me during the twelve months ending with April 1, 1908.[3] Of the

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[3] For a larger list of 217 cases previously operated upon by me, see Amer. Jour. Med. Sci., 1908, cxxxv, 137.

72 cases, 9 were examples of chronic, non-calculous cholecystitis, with no mortality; whilst of the remaining 63 cases of cholelithiasis, 4 were fatal.

Glancing over the histories of the 9 sufferers from non-calculous cholecystitis, I am impressed with the constancy of symptoms of dyspepsia, and the presence of adhesions. Thus, 7 of the 9 complained of such discomfort after eating, as oppression in the stomach, burning, and eructations of gas. And in 8 of the 9 cases adhesions were found, usually between the gall-bladder and some part of the alimentary tube, as the stomach, the duodenum, or transverse colon, or between the gall-bladder and omentum. These adhesions are relics of bygone infection, and make dyspeptics of their bearers. That the infection, as a rule, had long since departed was shown by positive cultures being obtained from only 2 of the 9 cases, while in 2 others there were no bacteria, but lymph nodes were enlarged, in one case along the cystic, and in another along the common duct. Furthermore these patients were subject to frequent attacks which increased in number with the duration of the disease. Such attacks usually followed dietary indiscretions, and started with pain in the right hypochondrum, radiating to the back and right shoulder blade, with nausea and vomiting, sometimes chills and fever, and sometimes followed by jaundice. Some patients complained of continuous tenderness over the gall-bladder, frequently aggravated by exercise —further evidences of the baneful effect of adhesions. The gall-bladders were usually atrophied, with walls thickened sometimes to as much as one-quarter of an inch. One patient had been operated upon two years previously, when 200 stones were removed from the gall-bladder. Within the past year he had three attacks of paroxysmal, crampy pain in the epigastrium, referred to the right scapula, preceded by chills and followed by gaseous eructations and vomiting. Operation revealed a greatly thickened gall-bladder, firmly adherent to the anterior abdominal wall. This gall-bladder was removed. Of the 9 cases I was able to save the gall-bladder in 6, which were merely drained; the remaining 3 were so badly diseased as to demand removal.

While cholecystitis in its various forms may be present without cholelithiasis, yet the two are so frequently wedded etiologically and pathologically that it is not strange that a differential diagnosis cannot always be made by clinical signs alone. The natural history of gallstones is interesting; their accurate diagnosis, satisfying; their removal, comforting.

Normal bile has been found contaminated with bacteria, but this is of little moment so long as there is no disease of the gall-bladder walls, and the biliary stream is unimpeded. Obstruction to the outflow of bile from this bulb, therefore, is the first step toward calculus formation, since whenever bacteria are bottled up they tend to create mischief, although now and then they die out.

Infection is the forerunner of inflammation; inflammation means cell proliferation, exudation, and altered function—a succession of events favorable to the precipitation of biliary salts. By means of mucin these salts are agglutinated, and with the cholesterin formed by degenerative changes in the epithelial cells, form calculi. At first the stones are soft, but, with time, become harder and harder. Recent soft calculi are preferred by bacteria to older, harder ones; for, whereas bacteria are continually present in the former, they are frequently absent from the latter. In a recent article on the etiology of cholelithiasis, Funke says: "While the almost constant presence of bacteria in recently formed calculi appears to indicate their presence in the bile at the time of the calculus formation, the experiments of Gilbert and Fournier and of Chauffard furnish sufficient ground to refute such conclusion. These investigators have succeeded in demonstrating secondary penetration of the calculi. They also succeeded in demonstrating that the organisms travel in the other direction. These observations may explain the frequency with which soft calculi are inhabited by organisms. The finding of sterile bile and infected calculi at the same operation can scarcely be held to controvert secondary penetration, for the bile may have become bacteria free after having infected the concretions. Although secondary penetration cannot be doubted in the light of the experiments quoted, yet there are few authors who hold that the presence of bacteria in the concretions must in all instances be regarded as such."

Without discussing further how gallstones become infected with bacteria, the fact remains that such bacterial hives argue ill for their bearer. Once formed, how long before gallstones produce wailing and gnashing of teeth? Richardson says: "During that more prolonged period of time between the first aggregation of the crystals which form the gallstone and the development of a gallstone large enough to obstruct the duct, a very considerable interval of time must elapse. Just how long that period of time may be is a matter of inference. Before they begin to offend, gallstones usually have a very considerable age. The age of a stone is judged not by its size, but by its consistency. A soft stone, easily crushed between the the fingers, of a light lemon color, I usually regard as a recent one. A dark, rough stone, especially if it is made up of different kinds of salts varying in color and in hardness, would probably be much older than a stone composed of salts of the same consistency and the same color."

I believe there is scarcely anyone with large experience in gall-bladder surgery who does not see examples of the different stages through which gallstones pass during their development, did he but recognize them as such. In discussing the pathology of cholecystitis, McFarland says: "Sometimes gall-bladders are found filled with inspissated bile, rich in bacteria, sometimes filled with bile containing a pulpy mass, probably a precipitate of the bile salts about this bacteria mass; sometimes the entire contents of the organ will be pasty, mushy, gritty, or gravelly; sometimes a single stone and sometimes a large number of stones will be present."

From the above remarks I think I am justified in bringing up the question of the preventive treatment of cholelithiasis. This, however, seems premature at the present time, since the early symptoms of even fully matured gallstones are just beginning to be understood and are not always detected; the symptoms that may be interpreted as forerunners of those of fully formed calculi are even less known.

More significance, I think, must be attached to the contents of opened gall-bladders. When a surgeon drains a gall-bladder of the products of cholecystitis; when he empties it of inspissated bile rich in bacteria, or of bile containing a pulpy mass; or when he evacuates pasty, mushy, gritty, or gravelly material, is he, with his knowledge of the natural history of gallstones, not justified in believing that in each of these instances he has carried out the preventive treatment of cholelithiasis? When he removes stones, since these are found after an unknown, but probably long lapse of time, he is operating comparatively late in the disease. And when he must perform choledochotomy for calculi that have reached as far as the common duct, or when he drains a gall-bladder for chronic pancreatitis that has resulted from gallstone disease, or removes tissue made cancerous by the same cause, he may find consolation in the old saying, "Better late than never."

Gallstones lodged in the fundus of the gall-bladder may be likened to shot in the breech of a gun, and the biliary ducts to the barrel of the gun. If the gallstones remain quiescent, as is claimed for them in a certain percentage of cases, the gun is never discharged. If, on the other hand, infection or some other factor starts the gallstone or stones along the biliary tract, the gun is discharged and the explosion manifests itself in the colicky pains and other disturbances the patient experiences. The missile or gallstone may travel *per vias naturales*, and drop harmlessly out of the anus, or be vomited forth. Or, by ulcerating a passage for itself, it has the effect of a gunshot wound from a missile of extremely low velocity, and makes its way by fistulæ directly to the surface, or into the digestive tube, or elsewhere.

It is doubtful if gallstones can be present without producing symptoms, as is claimed for about 5 per cent. of gallstone subjects. We now know that it is not necessary for a diagnosis that the manifestations of gallstone disease be displayed on transparencies bearing, in large letters, the words "colic" and "jaundice." We look to the earlier writings of the disease, and attach due significance to the various expressions of "indigestion," as epigastric distress, anorexia, flatulence, eructations, heart burn, nausea, and vomiting. If there is not out-and-out pain, there may be, as Richardson says, a consciousness of discomfort in the epigastrium, in the right hypochondrium, or sometimes in the left—"a discomfort for which the patient seeks some simple remedy which she has in the past found efficacious—a hot water bottle, a liniment, an aromatic, a digestive, a carminative." In such cases pericholecystic adhesions or gallstones provoke these slight symptoms. Richardson writes: "One patient examined very recently, who has a gall-bladder filled by two large stones, says that she has never had any trouble, but she goes on to say: 'Of course I have to be very careful what I eat on account of these bilious attacks which I have been subject to for years.'" Thus, not only must the patient restrict his appetite, a matter which means mental suffering even for the healthy, but he must suffer the consequences of dread of eating, which, continued for a long time, brings loss of weight and strength, and consequent weakness.

Of the 72 patients with gallstone disease reviewed for this article, 63 had gallstones, and of these 4 died. The stones were distributed as follows: 44 times in the gall-bladder; 5 in the gall-bladder and cystic duct; 1 in the cystic duct alone; 3 in the gall-bladder and common duct; 1 in the gall-bladder and common and hepatic ducts; 1 in the cystic and common ducts; 4 in the common and hepatic ducts; and 4 in the common duct alone. For practical consideration we may divide the stone-bearing areas into two groups, placing in the first group all stones that were found between the fundus of the gall-bladder and the termination of the cystic duct; and, in the second, all stones found in the hepatic and common ducts. Of the 63 patients, 12 were males and 51 females. Most of the patients were between the ages of forty and sixty years. Nine patients had had enteric fever, but the typhoid bacillus was recovered from only 2, both of whom had experienced the disease within six months previous to operation. The typhoid bacillus was reported from cultures of 2 others, neither of whom had ever had enteric fever. The colon bacillus was found in 11, and Staphylococcus aureus in 3.

Of the 63 patients, the disease had existed one week or less in 8; one year or less in 21; from one to five years in 16; from six to ten years in 7; from eleven to twenty years in 6; from twenty-one to thirty years in 1; from thirty-one to forty years in 1; and the duration was not mentioned in 3. Chronic dyspepsia was the complaint of 32. The pain was sudden and sharp, or crampy or spasmodic, and located over the gall-bladder in 57, but dull and burning in 1. It was referred to the back in 6, to the right scapula in 28, and to both back and scapula in 9. There was tenderness over the gall-bladder in 44; over Robson's point in 9. Rigidity of the right rectus was present in 9 cases. Attacks came on at night in 16. Nausea was the complaint of 2; vomiting of 20; and both nausea and vomiting of 22. Chills were present in 10 cases; chills and fever in 4; chills, fever, and sweats in 14; chills and sweats in 1; and fever and sweats in 1. Jaundice was present alone in 27 cases; jaundice with clay-colored stools and biluria in 10; while but 2 complained of itching of the skin from jaundice; 15 patients had lost from ten to sixty-five pounds in weight; 4 patients found stones in the stools. The liver was enlarged in 16. The gall-bladder was determined enlarged clinically in 6. The stomach extended down near or to the navel in 5. Carcinoma of the liver was seen once.

Adhesions were found as follows: around the gall-bladder in 4; around the common duct in 4; gall-bladder and stomach, 10; gall-bladder and liver, 1; gall-bladder and colon, 2; omentum and gall-bladder, 8; omentum and liver, 3; omentum, stomach and colon to liver, 4; obliterating the foramen of Winslow, 1; omentum, stomach and colon to gall-bladder, 1; gall-bladder, liver and stomach, 1; omentum, colon and pylorus, 1; omentum, colon, pylorus and anterior parietal peritoneum, 1; liver and anterior parietal peritoneum, 1; omentum, colon and gall-bladder, 1; omentum, gall-bladder and duodenum, 1. A pericholecystic abscess was found once. The gall-bladder was small in 4 cases; thickened in 9; both small and thickened in 9; distended in 8; and both thickened and distended in 11. Hydrops was present in 4 cases; empyema in 3; and gangrene in 1. The gall-bladder was involved by acute cholecystitis in 1 case; by chronic in 7; by chronic with acute exacerbation in 20, of which 3 showed in addition necrosis, and 1 was hemorrhagic and œdematous. The common bile duct was thickened and dilated in 2 cases. In 2 instances the gall-bladder showed a diverticulum. The stones ranged in number from 1 to 213, and were variously described as yellow or black, round or oval, facetted or mulberry. Gravel was present in 1 case. The quantity of bile varied from a few c.c. to 50 c.c. and was clear or cloudy, reddish, greenish-yellow, or black, and viscid or inspissated. Enlarged lymph nodes around the cystic duct were palpated in 2 cases.

As regards the operative procedures, cholecystectomy was performed 30 times; cholecystostomy, 24; cholecystotomy, 1; and choledochostomy, 19. Drainage was freely used, a rubber tube being placed in the gall-bladder in 24 cases; in the cystic duct in 13; in the common duct in 13; and in the hepatic duct in 6. Supplementary dossils of gauze insulated by either a split rubber tube, or else by rubber-dam, reënforced the primary tubes in 36 cases, and were placed in the fossa of the gall-bladder in 25; in front of and behind the right edge of the lesser omentum in 16; and in the subhepatic region in 18. Examination of the blood revealed hemoglobin percentage between 50 and 70 in 19 cases; from 71 to 80 in 21; and from 81 to 96 in 22. Leukocytosis varied between 3400 and 10,000 in 36 cases; from 10,000 to 20,000 in 19; and from 20,000 to 40,000 in 3. The coagulation time of blood was five minutes or less in 34 cases; and from five to fourteen minutes in 19. In some few instances no blood report was received. Differential leukocyte counts, and examinations of stomach contents and of feces were made with few exceptions.

Interesting pathological features were met with as follows: A spontaneous cholecystogastrostomy, separation of which revealed an opening in the stomach the size of a goose quill, necessitating gastrorrhaphy. In another case the pancreas was slightly enlarged, and a few hard areas were palpated in it; from this patient 13 gallstones, the size of a pea, were removed from the common duct. In another patient, a woman, aged forty years, tissue hyperplasia around the fundus of the gall-bladder, invading the liver for from one-quarter to one-half inch, was seen at operation; the pathological findings revealed, in addition to chronic calculous cholecystitis with acute exacerbation, marked proliferation of the mucous membrane of the gall-bladder, enough to warrant a tentative diagnosis of adenoma. One intrahepatic gall-bladder was found, deeply embedded in the liver, firmly contracted upon contained stones, and shrunken, distorted, and thickened to such extent that certain landmarks were obliterated; a very large, square, faceted stone had partially ulcerated through the fundus of this buried gall-bladder. In 2 cases cholecystectomy was performed because of a distinct diverticulum in the gall-bladder of one, and of a diverticulum near the junction of the cystic with the hepatic duct in another. A gall-bladder that contained 213 stones had to be ablated because some stones were embedded in its mucosa. Another gall-bladder was removed because a stone that blocked the cystic duct could not be dislodged. In this case, several whitish, circumscribed, elevated areas, one inch square, and supposedly carcinomatous, were seen upon the visceral surface of the liver. In a case of cholecystostomy, in addition to a rubber tube with gauze being placed into the gall-bladder, this organ was attached to the anterior parietal peritoneum to meet the indication for prolonged drainage. In another case while cholecystostomy was performed, yet the rubber tube could not be invaginated into the cavity of the organ on account of the thickness of the gall-bladder mucosa.

A few of the many complications of gallstone disease are illustrated in this series. In 2 cases in which the stomach was dilated to a finger's breadth above the navel, there were found numerous adhesions between the gall-bladder and pylorus. Ulceration of the gall-bladder was seen in the case of spontaneous cholecystogastrostomy, and in the case of intrahepatic gall-bladder, in which a stone was caught worming its way through the gall-bladder wall. The adhesions and pericholecystic abscess, the empyema and gangrene of the gall-bladder, and adenomatous degeneration of its mucosa, the chronic pancreatitis and carcinoma of the liver have all been referred to. Empyema of the chest and pneumonia of the right lung were fatal complications in one case. In a patient who had been jaundiced during the ten days previous to operation, from chronic obstruction of the common duct, the liver extended almost to the navel, and was tender—conditions sufficiently accounted for by finding 31 stones in the common duct, one of which was the size of a hickory nut, another in the ampulla, and 4 more in the left hepatic duct. Chronic appendicitis complicated one case of cholecystitis and 3 of cholelithiasis. I am not prepared to say in these 4 cases whether the appendix was primarily diseased and infected the gall-bladder secondarily through the portal system, or whether the infection extended from the gall-bladder to the appendix. In no instance was there impaction of a gallstone in the appendix, and this was certainly impossible in two of the appendices removed, since both were affected by chronic obliterative appendicitis. It is interesting to observe that a feature common to all 4 of these cases was prolonged indigestion, and this is noteworthy also because there were active simultaneously two of the most frequent causes of this malady, namely, chronic gallstone disease and chronic appendicitis.

Because of its frequency (about 15 to 20 per cent. in my cases), and because of the dire consequences when not removed, I shall say some few words on common duct lithiasis. In 29 out of 182 of my cases stones were found in the common duct and elsewhere, and merely to have removed the stones that were situated elsewhere, thereby overlooking those in the common duct, would have boded ill in any of the 29 cases. In the series of 63 cases of cholelithiasis now reported, stones were found in the common duct in 13. If these stones were not removed, nature might relieve the situation on behalf of the patient by the subsequent permanent external biliary fistula, mutely rebuking the surgeon for his bungling job, and patiently awaiting the aid of re-operation.

Although theoretically infection of the common duct directly from the intestine may be said to be resisted by the oblique intramural segment of the duct, and the presence of the terminal sphincter of Oddi, yet it is obviously impossible to say in how many people these supposed natural barriers have been prophylactic. It is true that the gall-bladder is infected by way of the common duct, though not so frequently as by the portal blood, and this organ is the primary focus for lithiasis of the common duct. While stones in the gall-bladder alone create much mischief, yet here the mischief is confined to a branch only of the biliary tract; but when stones pass out into the common duct and cannot pass through it the main line becomes blocked, and the entire biliary tract more or less involved. Stones in the common duct choke up more or less completely the total excrementitious filtrate of the whole liver, and as this filtrate contains whatever bacteria the portal blood could collect and the liver could not annihilate, the well-known severity of the symptoms of choledochus obstruction can thus readily be accounted for.

When acute this obstruction is complete, but the duct soon dilates from inflammation of its walls and *vis a tergo* of the bile, so that the obstruction becomes incomplete, or chronic, and the stone or stones are now at liberty to carry on the ball-valve action described by

Fenger. Perfectly in accord with the simple mechanical action is the behavior of the jaundice. At first rapidly reaching a deep color from acute complete obstruction, it slowly lifts as the duct dilates to allow partial drainage of the blocked bile, and it alternates at the will of the ball-valve stone. This ball-valve action is significant in several ways. In the first place, it is nature's method of dealing with an emergency by permitting drainage of the pent-up toxic fluids from the biliary vessels into the intestine. Then by variations in the jaundice it as much as tells the surgeon that the ailment is benign, for the jaundice of malignancy develops slowly and progressively and remains for all time. Finally, the ball valve helps to maintain Courvoisier's law by preventing enough backward pressure to dilate the gall-bladder.

Courvoisier's law is, of course, of no use clinically, but often affords a beacon light when the abdomen is entered. If there is chronic jaundice depending upon obstruction of the common duct, a shrunken gall-bladder is significant of calculous blockage of the choledochus, but a dilated gall-bladder indicates some other cause for the obstruction. This shrinkage of the gall-bladder is favored by the ball-valve action of the stone or stones as explained above, and is determined by inflammatory fixation of the contracted organ. Recently, M. R. Barker, of Chicago, has expressed the belief that Courvoisier's law is dependent upon the folds of Heister in the cystic duct: that in calculous obstruction of the common duct the normal action of these folds is not disturbed, thereby preventing dilatation of the gall-bladder by keeping the cystic duct closed; but in obstruction of the duct by pressure of a tumor, the diseased condition has involved either the folds of Heister or the nervous mechanism that controls them, or both, and their normal action is suspended or destroyed, allowing distention of the gall-bladder with bile and its own secretion.

In addition to the ague-like paroxysms of jaundice, colicky pain, and fever with the characteristic "steeple-chart" rises of Moynihan, subjects of choledochus obstruction are very apt to suffer loss in weight, a feature ascribed by Fenger, to "intermittent, frequent, ptomaïne intoxication—that is, bile absorption—as well as to disturbed digestion" (Moynihan). So, too, associated pancreatitis is apt to be another factor. This loss of flesh must be borne in mind when questions of malignancy arise.

Illustrative of the above remarks is the following case: Mrs. S., aged fifty-nine years, had been a sufferer from gallstone disease for seven years, during which time she had had innumerable attacks similar to, but not as severe as, the present. Three months before operation she was afflicted with severe, colicky pain in the right hypochondrium, radiating to the back and right shoulder. The pain was followed by vomiting, and this by chills, fever, and sweats. Three weeks before operation the pain became very severe, the attacks more frequent, and the jaundice very deep, with clay-colored stools. During the three weeks between this attack and the operation there was very little pain, and the jaundice began to clear, but the skin was very itchy. During these attacks the patient lost nearly 50 pounds in weight.

Examination showed universal jaundice and tenderness over the liver. Anemia to the extent of 68 per cent. of hemoglobin was present, and the blood coagulated within seven minutes.

Operation revealed in addition to adhesions between the omentum, pylorus, colon, and the gall-bladder, a small contracted and thickened gall-bladder free of stones. The common duct was much dilated, being as large as the little finger. A calculus the size of a marble was removed from the ampulla of Vater, and two smaller, irregular calculi from the hepatic duct. Drainage of the common duct was effected by a rubber tube, and of the sub-hepatic region and area in front of the lesser omentum, by Mikulicz drains.

By no means, however, will all cases be found so typical. In the absence of impaction no jaundice whatever may develop, and no severe pain arise. Endless modification of symptoms may be met with. Nevertheless, even in the absence of jaundice the common duct should always be determined free of stones.

In recent years pancreatitis has been recognized as a complication of gallstone disease. A small stone impacted in the ampulla of Vater may convert the bile and pancreatic duct into a single channel, thereby causing retrojection of bile and acute pancreatitis from irritation of the bile salts (chemical pancreatitis) and from the accompanying infection. Chronic pancreatitis may arise from extension of the inflammation from the common duct into the pancreas through the duct of Wirsung. Robson found pancreatic catarrh or interstitial pancreatitis in more than half his cases of common duct lithiasis. This is somewhat higher than the experience of W. J. Mayo who stated recently that in 268 operations on the common and hepatic ducts the pancreas showed disease in 18.6 per cent. I have found chronic pancreatitis in about 11 per cent. of my fatal cases of cholelithiasis. I report on these fatal cases, because in the living the diagnosis of chronic pancreatitis by palpation is very fallacious. As Richardson recently stated: "I would therefore exclude from all consideration of scientific accuracy my own cases, at least of so-called chronic pancreatitis, for their existence has never been proved by section, by microscope, or by autopsy. Palpation in these cases should always be directed first to the triangle of pancreatic inflammation, recently described by Mayo. This triangle is that part of the head of the pancreas which lies between the duodenum on the right of the ducts of Santorini above and Wirsung below. It is here that the organ is first infected."

If a stone in the common duct compresses the duct of Wirsung, the duct of Santorini will maintain pancreatic excretion in nearly half of the cases (Mayo). Letulle and Nattan-Larrier found that in 10 per cent. of patients the common bile duct and the duct of Wirsung empty into the duodenum by separate orifices, and it might be supposed that in this way the pancreas would be exempt from involvement. This is true in so far as chemical pancreatitis is concerned, but inflammation could yet spread from the common duct into that of Wirsung by travelling along the small extent of duodenum between them, and this, perhaps, explains the pancreatic involvement cited in New's case (cited by Egdahl), in which gallstones were present, but the duct of Wirsung had a separate opening. In this connection it would be interesting to learn if the pancreas is ever infected from the duodenum through the duct of Santorini when this is present. Opie has well said that in this unfortunate association of terminal facilities the large percentage of known diseases of the pancreas have their etiology (cited by Mayo).

I have recently operated upon two cases of acute suppurative pancreatitis, both of which showed fat necrosis. Both cases were drained retroperitoneally, and both recovered. In only one, however, was associated gallstone disease found. Furthermore, pancreatic cysts also may arise from pressure of a gallstone upon the duct of Wirsung.

It is my routine practice in all cases of gall-bladder affections, in addition to examination of the blood and stomach contents, to have the feces examined. Fatty stools in the absence of jaundice are very significant of chronic pancreatitis. If the urine shows glucose, and if it yields the acicular crystals of Cammidge's test, the diagnosis of pancreatitis is strengthened. My experience with Cammidge's test has been limited to the latest two cases of suspected pancreatitis admitted in hospital. In the first case the test was positive; in the second, it was positive at the first trial, but negative at the second. Of these, one did not have the disease, and if the other patient had pancreatitis, no opportunity of proof by operation could be had.

The medical treatment of gallstone disease may be disposed of in a few words. The task of dissolving gallstones or absorbing adhesions by medical means may be conservatively estimated as about equal to the twelve labors of Hercules, or the recurring toil of Sisyphus. The Carlsbad treatment is very useful to hasten convalescence from gallstone operations, and is a good place for those who, in the absence of contra-indications, refuse to be operated upon, since it promotes mental peace.

I shall speak somewhat more fully on the surgical treatment. In my experience the cases to which the prophylactic treatment for gallstones could be applied were those referred to me early by physicians. In such cases the contents of the gall-bladder showed the formative elements of undeveloped gallstones, and the draining away of such material usually resulted in cure. After gallstones have formed the risk of operation is the risk of delay, as I think I have shown throughout this paper.

My operative technique has recently been described[4] so that it is unnecessary to repeat it now. The principles that govern me in operations on the biliary passages are, in the first instance, those common to all abdominal operations, namely, to get in the abdomen as quickly as possible, to remain there as short a time as possible, and to get out as soon as possible, doing only that which seems necessary to restore the patient to health. I take pains in the proper disposal of packs, so as to wall off the operative field as completely as possible. Adhesions are avoided when they do not interfere with the proper execution of the operation. The biliary tract is searched for stones, and all that are encountered are removed. Free and sufficient drainage is instituted according to the requirements of each individual case. Postoperative treatment must be watchfully and intelligently supervised.

I do not favor cholecystotomy or choledochotomy without subsequent cholecystostomy or choledochostomy, and I believe that with the elimination of these two procedures the statistics of postoperative peritonitis would be considerably reduced.

Cholecystectomy is a procedure I avoid when possible. The gall-bladder should not be removed unless we feel that in its ablation we are taking away a menance to the future health and comfort of our patient. Sidney Phillips has well said that it is impossible for the surgeon to devise so perfect an automatic mechanism for storage and intermittent distribution of bile as the gall-bladder. Aside from the danger of hemorrhage attending cholecystectomy, we must look into the patient's postoperative future. If a second operation should at any time be required, we may be only too glad that we have previously preserved the gall-bladder as a valuable agent for drainage of the biliary tract, and that the operative field is comparatively free of adhesions, as is not likely to be the case if the gall-badder has been removed.

ASCITES IN TYPHOID FEVER.[1]

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Peritoneal effusion in typhoid fever, apart from peritonitis, must be of rare occurrence, as there is no reference to in it the literature on the subject. During the last three years six cases presenting

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[4] Surg., Gynec., and Obstet., January, 1908, 61.

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 12 and 13, 1908.